NASA Technical Memorandum 4444

A Proposed Performance Index for Galactic Cosmic Ray Shielding Materials

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National Aeronautics and
Space Administration
Office of Management
Scientific and Technical
Information Program
1993

Abstract

In past studies, the reductions in absorbed dose and dose equivalent due to choice of material composition have been used to indicate shield effectiveness against exposure to galactic cosmic rays. However, these quantities are highly inaccurate in assessing shield effectiveness for protection against the biological effects of long-term exposure to the galactic heavy ions. A new quantity for shield performance is defined herein that correlates well with cell killing and cell transformation behind various shield thicknesses and materials. In addition, a relative performance index is identified that is inversely related to biological injury for different materials at a fixed shield mass and is directly related to the ratio of the fourth- and the second-order linear energy transfer (LET) moments.

Introduction

Conventional practices in radiation protection are considered adequate (ref. 1) against exposures in space to the relatively low linear energy transfer (LET, defined as a measure of radiation quality) radiations trapped in the magnetic field of the Earth. The establishment of a lunar base or the human exploration of Mars introduces the complicated problem of providing protection from the high charge and energy (HZE) particles of the galactic cosmic rays (GCR). (See ref. 2.) For these radiations, the reduction of biological risk due to the shielding properties of structural materials is uncertain. Further discussions of these issues are given in references 3, 4, and 5. At best, biological risk factors are estimated for these radiations only within a factor of 4 5, and the distribution of LET is known only within a factor of 2 3 for any given shield material (refs. 6 and 7). Hopefully, risk coefficient uncertainties will be substantially reduced in the next several years (ref. 2). At present, a materials research program is in progress to reduce uncertainty in the basic cross-sectional data and to optimize shielding properties through materials selection (refs. 5 and 8).

Because the biological response for the high-LET components is uncertain, a measure of "goodness" of a particular material type is difficult to formulate. Therefore, a quantitative statement of the shield effectiveness of a specific material cannot be presented until a fuller understanding of the biological response is available. However, if the biological risk can be separated into a biology-dependent factor and a factor based on the physical properties of the radiation fields, then the shield properties alone determine the physical risk factors. The biological factors must be investigated further by the radiobiologist in the future.

In this report, we attempt to separate physical and biological factors using basic concepts in microdosimetry. The physical factors are the moments of the LET distribution. The effects of the material properties on the moments are evaluated as a measure of shield performance. A GCR shield-performance index is proposed for materials characterization, which is closely related to the clonogenic death and neoplastic transformation of the C3H10T1/2 cell system (ref. 9).

Microdosimetry

The response of living tissue (ref. 1) to a dose D_γ with low LET is represented by a sensitivity coefficient k_γ and a quadratic coefficient D_o as

$$R_\gamma = k_\gamma D_\gamma (1 + D_\gamma / D_o) \tag{1}$$

where R_γ is either the risk of inducing a specific end point or the level of severity (ref. 10). The parameter D_o is dose-rate dependent and is on the order of 1.2 Gy for dose rates >50 mGy/day (ref. 1). We assume herein a low dose rate so that D_γ^2 may be neglected, where

$$R_\gamma = k_\gamma D_\gamma \tag{2}$$

Tissue cells are not all equal at low exposures because the energy deposits are quantized and energy is deposited in only a fraction of cells; similarly, volumes within a given cell are not all equally sensitive. In general, absorbed dose D is not a good measure of biological damage because an average quantity can be decomposed (ref. 11) as follows:

$$D = \frac{\sum \varepsilon_i}{V N_E} = \frac{\sum \varepsilon_i}{V N_H} \frac{N_H}{N_E} \tag{3}$$

where ε_i is the energy absorbed per hit cell, referred to as the hit size of the ith event, V is the cell

volume, and N_E is the number of exposed cells. At low dose, not all cells are hit, so the number of hit cells N_H is less than the number of cells exposed. Only as $N_H \rightarrow N_E$ is D meaningful in terms of tissue response (ref. 11). The fraction of cells that are hit at low exposure (that is, $N_H \ll N_E$) is

$$\frac{N_H}{N_E} \approx \sigma_g \phi \tag{4}$$

where σ_g is the geometric cross section and ϕ is the charged-particle fluence within the tissue system. Although the cross section can be larger than the geometric cross section due to the δ-ray diffusion, equation (4) is assumed herein to be a first-order approximation. The fluence ϕ is related to the absorbed dose D and radiation value of LET L as

$$\phi = 6.24 \frac{D}{L} \tag{5}$$

for ϕ in particles/μm^2, D in Gy, and L in keV/μm. For γ rays, L_γ corresponds to the secondary electrons generated and has a value of about 0.2 keV/μm; the corresponding ϕ_γ is an effective secondary electron fluence that is dependent on the photoabsorption coefficient and the γ-ray fluence. The distribution of hit size ε_i is approximately the continuous distribution $f(\varepsilon)\,d\varepsilon$, so the absorbed dose is written as two factors

$$D = \left(6.24\sigma_g \frac{D}{LV}\right)\left(\frac{\sum \varepsilon_i}{N_H}\right) \equiv 6.24\sigma_g \frac{D}{VL} \int \varepsilon f(\varepsilon)\,d\varepsilon \tag{6}$$

demonstrating that $0.16\,(VL/\sigma_g)$ is the average event size $\langle\varepsilon\rangle$ within the cell. This relation for average event size is the usual microdosimetric relation, where average lineal energy (lineal energy is the event size ε_i divided by cell mean chord) is numerically equal to LET (ref. 12). The fraction of cells that are hit and the mean event energy are shown for 1-Gy exposure in figure 1. The multihit region assumes Poisson statistics. The low-LET region ($L < 5$ keV/μm) involves exposure of a large fraction of the cells; however, few cells are directly irradiated at high LET ($L \gg 5$ keV/μm), and those cells that are hit receive large energy deposits (>100 keV). On this basis alone, we expect a substantially dissimilar biological response to 1-Gy exposure by radiations of greatly different LET. Yet the important factors in predicting tissue response depend on the probability of cell injury at a given event level, the efficiency of cellular repair, and the cell role in tissue function. As shown in figure 2, we have calculated the geometric hit frequency, the initial level of cell injury, and the unrepaired cell injury leading to clonogenic death in a C3H10T1/2 mouse cell population (ref. 13). Figure 2 shows that although the cell is most often hit by protons and helium ions, the probability of injury is small and the repair efficiency is high, with little permanent injury. Conversely, a high probability of injury and near-zero efficiency of repair occur from hits of silicon and iron ions. As a consequence, most clonogenic death from GCR exposure comes from ions with LET above 10 keV/μm (ions above carbon). Radiation injury from these ions shows minimal cellular repair. As a result, dose protraction (an extended exposure period) for GCR exposure will be less effective in reducing the biological response.

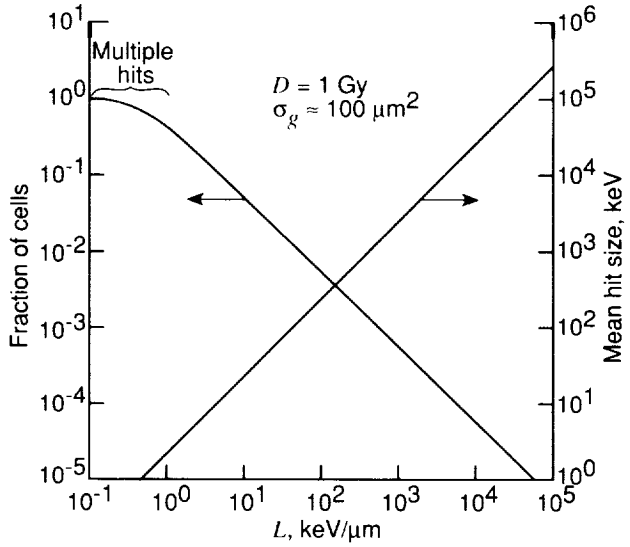


Figure 1. Fraction of hit cells and hit size as a function of linear energy transfer L.

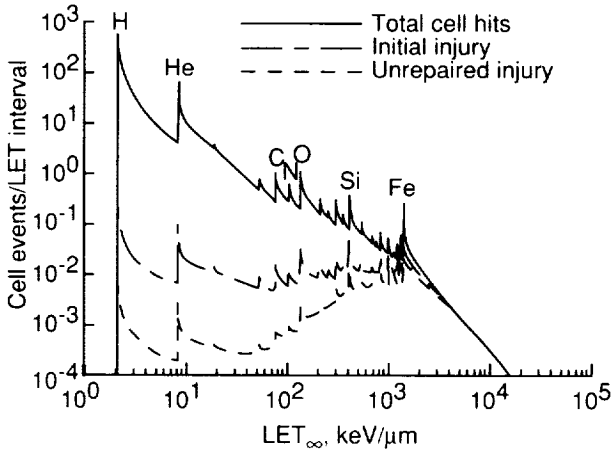


Figure 2. Cell events/log L interval for 1-yr exposure to galactic cosmic rays at solar minimum with 5 g/cm^2 of aluminum shielding.

Bond and Varma (ref. 14) postulate that the outcome of a specific event of size ε is represented by

the hit size effectiveness function $P(\varepsilon)$; therefore, the risk function for occurrence within a cell population is

$$R_L = \frac{N_H}{N_E} \int P(\varepsilon)\, f_L(\varepsilon)\, d\varepsilon = 6.24\sigma_g \frac{D_L}{L} \int P(\varepsilon)\, f_L(\varepsilon)\, d\varepsilon \tag{7}$$

for radiation of LET value L, with a corresponding hit size distribution $f_L(\varepsilon)$. Equation (7), as applied to cells, provides a clear separation of cell properties $P(\varepsilon)$ that incorporates the physiochemical processes used to achieve the end point. (See ref. 14 for further discussion.) For tissue applications, $P(\varepsilon)$ includes factors related to induction and promotion of the tissue system to the final, observed end point. The term $f_L(\varepsilon)$ is a physical quantity related to LET and the geometry of the cell. The term $P(\varepsilon)$ must be extracted from experimental response data for a sufficiently large dynamic range of $f_L(\varepsilon)$ spectra; customarily, $P(\varepsilon)$ is expanded as a series of terms with coefficients adaptable to experiments. In the present case, we use a power series to approximate the hit size effectiveness

$$P(\varepsilon) = \sum b_i \varepsilon^i \tag{8}$$

for which $b_0 = 0$ because $P(0) = 0$. Then equation (7) can be written

$$R_L = 6.24 \frac{\sigma_g}{L} D_L \sum b_i \langle \varepsilon^i \rangle \tag{9}$$

The first moment ($i = 1$) is linearly related to dose; the ratio of the second moment to the first moment is linearly related to average relative biological effectiveness (RBE) for low-dose neutron exposure (ref. 15) and is approximately related to the average quality factor Q in conventional practice (ref. 12). The higher moments are related to undetermined biological effects of very high LET events; however, even the use of a quality factor is uncertain (refs. 4 and 12). We assume that the limit of low LET is matched to the γ-ray response (eq. (2)) by

$$k_\gamma = 6.24 \frac{\sigma_g}{L_\gamma} b_1 \langle \varepsilon^1 \rangle \equiv V b_1 \tag{10}$$

and that equation (9) can be simplified (ref. 12) because $\langle \varepsilon^n \rangle \propto L^n$. In a mixed environment, the total risk R is the sum over all LET components

$$\begin{aligned} R &= 0.16 \int k_\gamma (L + a_2' L^2 + a_3' L^3 + \cdots) \phi_L \, dL \\ &= 0.16 k_\gamma \langle L \rangle \phi + 0.16 k_\gamma \sum_{i=2}^{n} a_i' \langle L^i \rangle \phi \end{aligned} \tag{11}$$

where ϕ is the total fluence. Within this microdosimetric model, the future clarifications of the biological response would correspond to new values for a_i'. Dosimetry for more conventional radiations would replace $1 + a_2' \langle L^2 \rangle / \langle L \rangle$ with Q (ref. 15) and would leave most higher terms undefined. For deep-space missions, the a_i' terms for $i > 2$ must be determined for the end points of interest as clarified by the biological experiments with very high LET radiations. In this sense, the validation of a shield code would ensure the accurate prediction of LET moments, and the preferable choice of materials would minimize the higher LET moments $\times$ Total fluence.

On the basis of conventional dosimetry, the shield material yielding the lowest value of $\langle L^2 \rangle / \langle L \rangle$ would minimize the dose equivalent, thus would be judged the "best" material. If conventional practice is adequate for the GCR environment, then the issue is quickly settled. However, if the higher order terms are important, then a moment for which $\langle L^p \rangle / \langle L^q \rangle$ when $p > 2$ and $q < p$ may indicate more closely the biological consequences. This question is addressed in the next section.

LET Distributions

Aluminum is the most common structural material used in space construction because of its strength per mass and good thermal properties. For this reason, aluminum will be presented as the standard of comparison herein. The LET distribution of galactic ions in free space and the attenuation of the ions as they pass through thicknesses of aluminum shielding are shown in figure 3. The LET values are for each ion in water. The distribution for 2 g/cm^2 corresponds to a basic pressure vessel wall with its micrometeoroid bumper, the distribution for 5 g/cm^2 corresponds to a moderately shielded compartment, and the distribution for 10 g/cm^2 corresponds to a heavily shielded vehicle. The main effect of the shield is to attenuate ions with LET above 7 keV/μm, which partially converts them into particles of lower LET. Note that the flux of carbon ions is largely unchanged. (See fig. 3.) The five lowest moments of LET are shown in table 1. The zeroth-order moment is the total particle flux in 1 yr; this value increases slightly with shield depth. The first-order moment is the locally absorbed dose in a water sample. The conversion factor is 1.6×10^{-10} Gy-cm/MeV. Although flux increases moderately, absorbed dose decreases as the aluminum shield depth is increased. The decreasing dose is related to an even faster decrease in average LET. The second-order moment ($i = 2$) is nearly proportional to dose equivalent; the factor 7.06×10^{-13} yields a quality factor of 7.5. Although

the absorbed dose decreases only slowly with increasing shield depth, the dose equivalent shows a substantial decrease related to the decreasing quality factor at larger shield depths. The average quality factor is nearly proportional to the ratio $\langle L^2 \rangle / \langle L^1 \rangle$, which is substantially reduced at larger depths. The higher moments show even greater decreases with shield depth and have not yet been connected to dosimetric-related functions. The behavior of the moments in other materials is qualitatively similar but, as will be shown, with important quantitative differences.

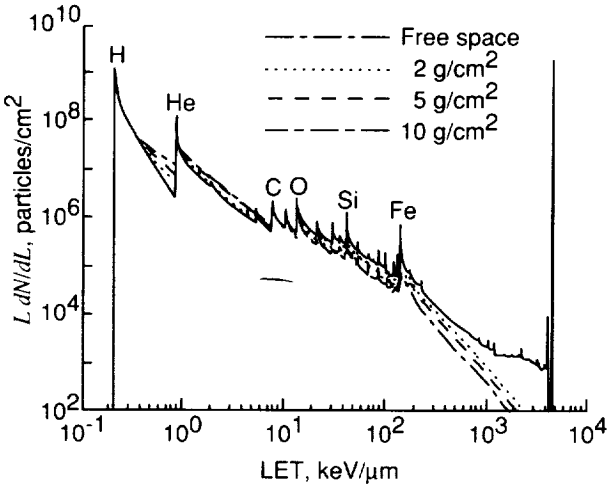
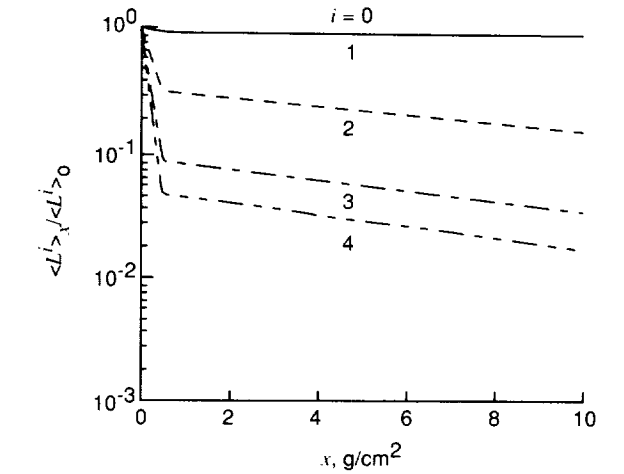


Figure 3. Annual particle flux/log L interval, shielded by various thicknesses of aluminum.

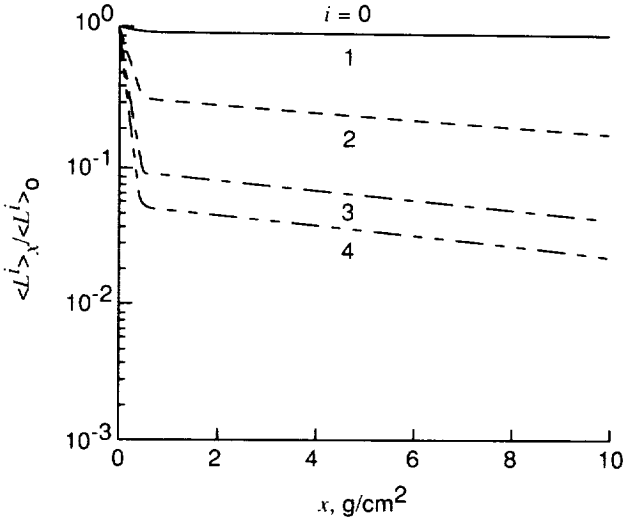
The attenuation of the moments as a function of depth are shown in figure 4. Aluminum, the standard space construction material, is shown in figure 4(a). Comparison of figures 4(b) and 4(a) indicates that iron is a slightly poorer shield material than aluminum. However, comparison of figure 4(c) with figures 4(a) and 4(b) shows that polyethylene is a greatly improved choice for shielding. On the basis of figure 4, polyethylene shows the best material properties of the three choices; however, assigning a quantitative advantage to this choice from the results in figure 4 is difficult.

In the past, dose equivalent H has been used to indicate biological risk for GCR exposures (refs. 1 and 7). Shield performance of a material m can be defined relative to aluminum shielding technology with the expression

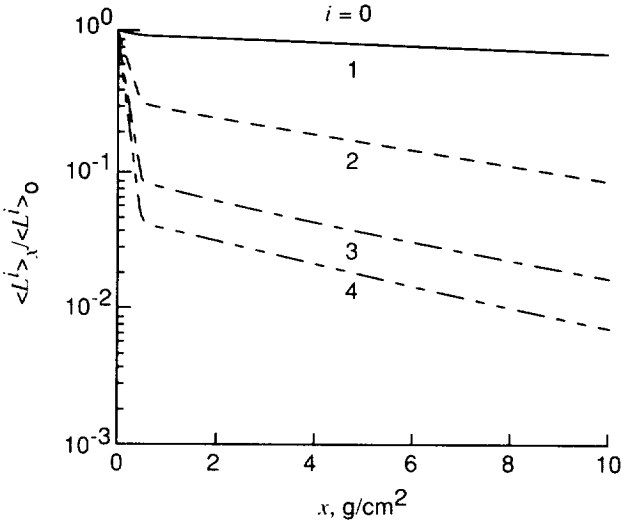
$$\text{Performance} = \frac{H_{\text{Al}}(x)}{H_m(x)} \qquad (12)$$



(a) Aluminum.



(b) Iron.



(c) Polyethylene.

Figure 4. Attenuation of linear energy transfer moments in diverse shield materials.

Table 1. Moments of LET Behind Shield Materials for 1-yr GCR Exposure at Solar Minimum

Areal density x, g/cm^2	Moments of LET, MeV/cm^2-cm^{-2} for i of				
	0	1	2	3	4
Aluminum					
Free space	1.29E+08	1.00E+09	1.70E+12	3.70E+16	1.18E+21
1	1.31E+08	9.18E+08	5.11E+11	3.08E+15	5.38E+19
2	1.32E+08	9.16E+08	4.70E+11	2.78E+15	4.84E+19
5	1.35E+08	8.97E+08	3.65E+11	2.01E+15	3.42E+19
10	1.38E+08	8.66E+08	2.53E+11	1.24E+15	2.05E+19
Iron					
Free space	1.29E+08	1.00E+09	1.70E+12	3.70E+16	1.18E+21
1	1.31E+08	9.43E+08	5.32E+11	3.32E+15	5.96E+19
2	1.34E+08	9.38E+08	4.93E+11	3.03E+15	5.41E+19
5	1.35E+08	9.42E+08	4.07E+11	2.35E+15	4.14E+19
10	1.38E+08	9.23E+08	3.02E+11	1.58E+15	2.72E+19
Polyethylene					
Free space	1.29E+08	1.00E+09	1.70E+12	3.70E+16	1.18E+21
1	1.30E+08	8.75E+08	4.65E+11	2.66E+15	4.46E+19
2	1.31E+08	8.49E+08	4.00E+11	2.20E+15	3.65E+19
5	1.33E+08	7.87E+08	2.61E+11	1.28E+15	2.03E+19
10	1.34E+08	7.16E+08	1.43E+11	5.86E+14	8.64E+18

where H_{Al} is the dose equivalent behind an aluminum shield of areal density x in g/cm^2 and $H_m(x)$ is the dose equivalent behind shield material m of the same areal density. Because the living space is a large container, the shield mass is approximately Ax, where A is the shield surface area (cm^2). Thus, performance in equation (12) is a measure of risk change by choice of material composition of the shield with a fixed shield mass. Thus, a material with a shield performance of 2 would reduce the dose equivalent by a factor of 2 without changing the shield mass. To test the validity of the performance index from equation (12), we compare the ratio of risk of clonogenic cell death of C3H10T1/2 cells behind an aluminum shield $D_{Al}(x)$ to the clonogenic death behind a shield of material m of the same areal density $D_m(x)$ in figure 5. We first note a nonlinear relationship between cell death and dose equivalent behind the shield materials considered. Second, a 10-percent increase in dose equivalent for an iron shield leads to a 20-percent increase in cell death. Conversely, dose equivalent always indicates an underestimation of the improved performance of polyethylene by up to 50 percent; therefore, dose equivalent is clearly a poor indicator of shield performance.

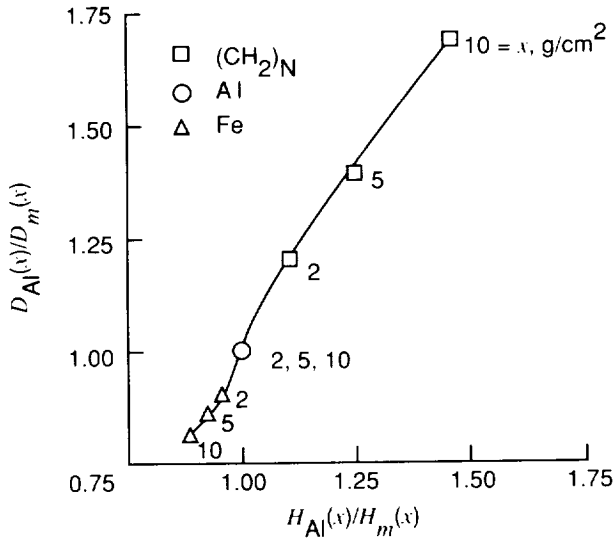


Figure 5. Relative cell killing as a function of relative dose equivalent of diverse shield materials of equal areal density x.

Proposed Shield Performance Index

To assign a quantitative measure of shield performance, we considered a track structure kinetics model of the C3H10T1/2 cell system for clonogenic death and transformation (ref. 13). Results of this model for a 1-yr exposure behind an aluminum shield with an areal density of 5 g/cm^2 is shown in figure 2. We have evaluated this model for the three shield materials used in the present study at areal densities

of 2, 5, and 10 g/cm^2 for each material. The depths in units of areal density (g/cm^2) are proportional to the total shield mass of a large shielded region. The conditions assume a stationary G_1-phase exposure for a constant dose rate over the 1-yr period. The cell death for an aluminum shield $D_{\mathrm{Al}}(x)$ of areal density x compared with the cell death for a different material $D_m(x)$ of the same areal density is expressed by

$$\text{Cell-death ratio} = \frac{D_{\mathrm{Al}}(x)}{D_m(x)} \tag{13}$$

This ratio measures the relative biological protection of the two materials. As shown in figure 5, the cell-death ratio is not well correlated with dose equivalent. A correlation of cell death was found in terms of the square of the ratio of the fourth moment $\langle L^4 \rangle$ to the second moment $\langle L^2 \rangle$

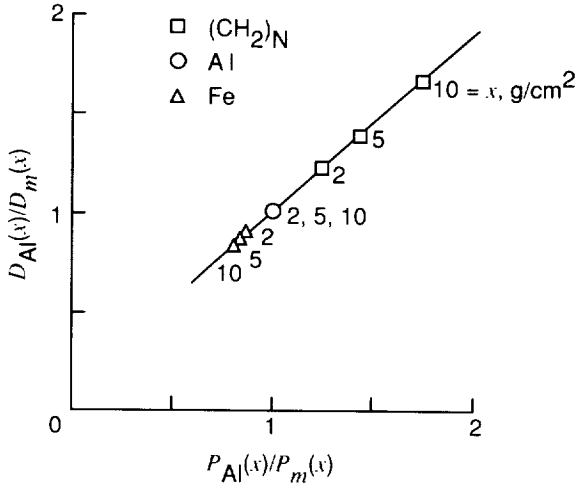
$$p_m(x) = [\langle L^4 \rangle / \langle L^2 \rangle]^2 \tag{14}$$

The cell-death ratio is shown in figure 6(a) with the relative performance index $P_m(x)$, which is defined as

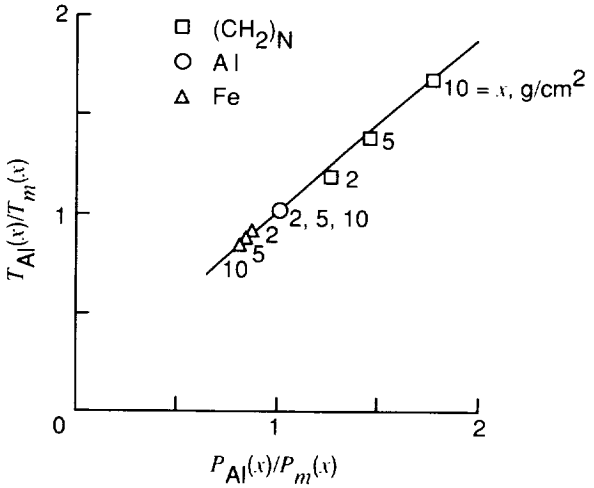
$$P_m(x) = \frac{p_{\mathrm{Al}}(x)}{p_m(x)} \tag{15}$$

for the data in table 1. Likewise, cell transformation $T_m(x)$ calculated for different shields is shown in relation to the shield performance index in figure 6(b).

Figure 6 shows that the relative performance index for aluminum as well as for cell-death ratio is unity by definition at all depths. The polyethylene values for areal densities of 2, 5, and 10 g/cm^2 are shown by the ray of points in the first quadrant for the center at (1, 1). The extreme point at 10 g/cm^2 has a relative performance index for polyethylene of $\approx$1.8, which corresponds to a cell-death ratio of $\approx$1.7. Similar results are obtained for transformations. Our interpretation is that the 80-percent increase in relative performance corresponds to a 70-percent decrease in biological injury. The excellent linearity of the cell-death and transformation ratios with relative performance index for all shield materials and areal densities validates the relative performance index of equation (14) as a metric of reduced biological risk (at least for the C3H10T1/2 stationary cell system). The simple interpretation that biological sparing is the inverse of the relative shield performance index might be used to evaluate shield worth. Thus, if $P_m(x) = 2$ for a given areal density x, then material m would provide approximately twice as much biological protection as the aluminum shield without increasing the shield mass. Although the relative performance index of polyethylene continues to increase for increasing shield mass, the relative performance of iron is only weakly dependent on shield mass beyond an areal density of 2 g/cm^2. Even though the relative performance index defined by equation (15) interprets the GCR environment reasonably well, whether this index will be equally useful for other environments remains to be seen.



(a) Cell killing.



(b) Cell transformations.

Figure 6. Relative cell killing and cell transformations as a function of relative shield performance index of diverse shield materials of equal areal density x.

Concluding Remarks

Although only modest changes are observed in the three lowest LET moments of the attenuated environment (corresponding to fluence and dose, and approximately to dose equivalent) for different materials at a fixed shield mass, the higher moments

show an increasingly strong material dependence, especially at the larger depths. The material dependence of cell killing and transformation is more characteristic of the higher than the lower LET moments, which demonstrates that dose and dose equivalent are poor indicators of biological risk for the GCR environment. The performance index introduced herein is directly related to the ratio of the fourth- and second-order moments of LET and inversely related to the increased biological sparing of the C3H10T1/2 cell system. This performance index relative to values for aluminum as a standard is proposed for evaluation of GCR shield materials.

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April 20, 1993

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REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1. AGENCY USE ONLY *(Leave blank)*	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED
	August 1993	Technical Memorandum

4. TITLE AND SUBTITLE
A Proposed Performance Index for Galactic Cosmic Ray Shielding Materials

5. FUNDING NUMBERS
WU 592-43-21-01

6. AUTHOR(S)
John W. Wilson, J. S. Wood, Judy L. Shinn, Francis A. Cucinotta, and John E. Nealy

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
NASA Langley Research Center
Hampton, VA 23681 0001

8. PERFORMING ORGANIZATION REPORT NUMBER
L-17199

9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)
National Aeronautics and Space Administration
Washington, DC 20546-0001

10. SPONSORING/MONITORING AGENCY REPORT NUMBER
NASA TM-4444

11. SUPPLEMENTARY NOTES

12a. DISTRIBUTION/AVAILABILITY STATEMENT
Unclassified Unlimited

Subject Category 93

12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*

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14. SUBJECT TERMS
Shielding materials; Nuclear; Galactic cosmic rays

15. NUMBER OF PAGES
8

16. PRICE CODE
A02

17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT
Unclassified	Unclassified		